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Incidence and risk factors for post-penetrating keratoplasty glaucoma

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Purpose: To carry out a prospective study to analyze the incidence and various preoperative, intraoperative, and postoperative risk factors for the development of PPKG. **Methods:** A total of 207 patients were analyzed prospectively, who were operated for penetrating keratoplasty (PK) in a tertiary eye care hospital between the time period of August 1, 2017 and February 28, 2018 and were followed up till the sixth month. Each patient was analyzed at every visit to determine the factors responsible for post-keratoplasty glaucoma. **Results:** Out of 207 eyes, post-PK glaucoma developed in 84 cases, which yielded an incidence of 41%. Incidence of PPKG (Post PK glaucoma) in various conditions was as follows: in repeat PK 62%, in perforated corneal ulcer 33%, in nonperforated corneal ulcer 29%, in corneal scar including adherent leukoma 37.2%, and in pseudophakic bullous keratopathy and aphakic bullous keratopathy, 14% and 80%, respectively. In age- and sex-adjusted multivariate analysis, the significant risk factors were age (*P*-value- 0.006), presence of PAS (*P*-value 0.001), and fellow eye glaucoma (*P*-value 0.04). Aphakia and combined surgery were not found to be significant. **Conclusion:** Our study recommends a meticulous examination of the fellow eye to assess the presence of glaucoma as it can increase the suspicion of glaucoma in the eye to be operated. The presence of PAS and age are important risk factors for developing PPKG. The risk of developing PPKG increases exponentially as the number of risk factors increases, but the presence of more than three risk factors does not add to the development of PPKG.

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Penetrating keratoplasty (PK) and endothelial keratoplasty are the most common types of keratoplasties. Despite technological advances in the fields of corneal preservation, surgical techniques, and postoperative care, complications after corneal grafting surgery are not rare. While some of these complications like graft infection and graft rejection threaten graft survival, others like high post-keratoplasty astigmatism and post-PK glaucoma prevent the achievement of optimal visual acuity even with a clear graft by causing irreversible damage to the optic nerve. The complexity of post-PK glaucoma lies in the inherent difficulty in detecting, monitoring, and treating the condition. Post-PK glaucoma refers to the presence of persistently elevated Intraocular pressure (IOP) above 21 mmHg or an elevation of 20% from the baseline, with or without visual field loss and optic nerve changes, which requires the introduction of glaucoma medical therapy or surgical intervention.[1] The incidence of glaucoma after PK reportedly ranges from 9% to 31% in the early postoperative period and from 18% to 35% in the late postoperative period.[2–7] Various risk factors have been studied to understand the mechanism of post-PK glaucoma. Lens status (aphakia and pseudophakia),[4] repeat keratoplasty,[8] peripheral anterior synechiae, combined surgery, suturing technique, and graft size, including other factors have been implicated for post-PK glaucoma.[4,8] Certain factors are modifiable; thus, understanding the timeline of these factors can help in developing the appropriate management protocol. The purpose of this study was to determine the risk factors for developing post-PK glaucoma in a prospective manner along with the role of the fellow eye in early detection of the disease, which has not been given much importance in published literature.

Methods

We conducted a prospective observational study at a tertiary eye care center. The study was approved by the Institutional Review Board and the Institutional Ethics Committee. All consecutive patients who underwent PK between August 01, 2017 and February 28, 2018 were included in the study. Written informed consent was taken and they were followed up for a minimum period of 6 months. This study adhered to the tenets of the Declaration of Helsinki.

Detailed clinical workup including the history of previous corneal transplant or any previous ocular surgery or steroid use was noted. Ophthalmic examination included visual acuity, slit-lamp examination, gonioscopy, and applanation tonometry. The preoperative vision assessment was done

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by Snellen's chart or Landolt's C chart (for illiterate people). Snellen acuity was converted to Logarithm of the Minimum Angle of Resolution (logMAR) acuity. IOP was measured by Goldmann applanation tonometry (GAT) wherever possible. Digital tension (DT) was checked by experts and was taken as inferred IOP when GAT was not possible. IOP was measured by the standard technique in the normal eye first and then digital palpation was performed on both eyes to get inferred IOP of DT. Normal DT was considered as inferred IOP of ≤21 mmHg, high DT as >21 mmHg, and low DT as ≤6 mmHg. Ultrasonography was done if the posterior segment was not well visualized on indirect ophthalmoscopy. Ultrasonography has been described in literature as a reliable surrogate tool that can be used in quantifying Optic nerve head (ONH) cupping in cases of media opacities which preclude optic disk visualization, although this cannot be considered as the best tool for disk evaluation.[9–11]

All PKs were performed by standard surgical technique under local anesthesia (general anesthesia for children).[12,13] Peripheral iridotomy, pupilloplasty, or vitrectomy was done wherever needed. Topical steroid (prednisolone) was instilled in the operated eye four times a day initially and the frequency was increased or decreased depending on the degree of inflammation. Topical antibiotics were given four times in the initial period. If there was a sign of graft rejection, intensive topical steroids were given. Topical glaucoma medical therapy (timolol 0.5%) was given twice daily wherever needed. Patients were followed up at postoperative day 1 and 1 week, 1 month, 3 months, and 6 months postoperatively. At every visit, evaluation was done for visual acuity, graft clarity, the status of corneal epithelium, sutures (tight/loose/infiltrate), anterior chamber depth, inflammation, and IOP. All the patients were referred for glaucoma consultation, and IOP measurement and disk evaluation were done. For the purpose of this study, we divided the patients into those with transient glaucoma, persistent glaucoma, and no glaucoma. Transient glaucoma was defined as patients whose IOP recording was >21 mmHg at any point of time during the study period and glaucoma medical therapy was prescribed to control the IOP. This was if the requirement of AGM was short-lived (<3 months) and there was no associated optic disk change or visual field abnormality recorded. Persistent glaucoma was defined as raised IOP at any point of time during the study period requiring AGM throughout the follow-up period of 6 months or when glaucoma surgery was needed or patients with glaucomatous optic disk on fundus evaluation with or without significant cupping noted. No glaucoma status post-PK was defined as IOP persistently <21 mmHg, no requirement for glaucoma medication/laser/surgical procedure, or lack of evidence of optic disk or visual field changes suggestive of glaucoma during the period of the study.

In the case of early rise of IOP after PK, the patient was started on aqueous suppressants with β-blocker as the first line of AGM. Persistent elevation of IOP in the early postoperative period required either Neodymium-doped yttrium aluminium garnet (Nd:YAG) iridotomy in case of pupillary block or AC wash if hyphema/lens particles were found to be the cause. In the presence of open angles, steroid-induced glaucoma was considered and steroids of low potency were used. In case the angles were found to be closed, the extent of Peripheral anterior synechiae (PAS) was noted and medical therapy was started with β-blockers as a first line. Oral Carbonic anhydrase was administered for a short duration to achieve immediate lowering of IOP, taking care of the graft status. In case of high IOP not controlled on maximum medication, surgical management was the treatment of choice.

Results

A total of 215 PKs were done during the study period. Two hundred and seven eyes of 207 patients were taken up for data analysis (eight patients were excluded due to loss of follow-up). The mean age of the patients was 47.86 ± 19.94 years (range 2–90 years). Male recipients were more (71%) than female recipients (29%). Optical PK was more commonly performed (60.87%) than therapeutic PK (39.13%). Of the 207 patients, secondary glaucoma developed in 149 patients, of whom 65 (31.40%) patients had a transient rise in IOP and 84 (40.58%) patients developed persistent glaucoma [Table 1]. Development of post-PK persistent glaucoma was found in 62% of the failed graft cases, 43% of eyes with a corneal scar [Fig. 1a and b], 33% of perforated corneal ulcer, 29% of nonperforated corneal ulcer, 27% of adherent leukoma, 80% of eyes with aphakic bullous keratopathy, and 14% of Pseudophakic bullous keratopathy (PBKs) [Table 2]. Distribution of patients according to the presence of risk factors has been enumerated in Table 3.

The degree of association between the risk factors and post-PK glaucoma was measured by odds ratio. The odds ratios for individual factors were first estimated by Fisher exact test. Subsequently, four binary logistic regression models were fitted with the dataset in order to estimate the odds ratios for individual factors [Table 4].

One of the preoperative risk factors identified was age > 50 years. 47.2% of patients >50 years of age developed persistent glaucoma ($P = 0.049$), which was also found to be significant in logistic regression analysis (P-value 0.006). Fifty-three patients were diagnosed to have preoperative glaucoma, of whom 31 patients (58.5%) developed persistent glaucoma. This was found to be a significant risk factor in univariate analysis (P-value 0.002); but in regression analysis, it was not found to be significant. Repeat PK for failed graft was performed in 47 patients, and 29 (61.7%) patients developed persistent glaucoma. This was found to be significant in univariate analysis (P-value 0.0008) as well as multivariate analysis (P-value 0.008). Preoperative diagnosis of glaucoma in eyes with a corneal scar, corneal ulcer, or any media haze becomes difficult. In such situations, disk evaluation of the fellow eye was done and it was observed that 14 patients had glaucomatous disk changes. Of these 14 patients, 11 (78.57%) patients developed persistent glaucoma after keratoplasty in the operated eye and three (21.43%) patients had a

Table 1: Distribution of glaucoma

Glaucoma	No. of patients	Percentage
No glaucoma	58	28.02
Transient rise in IOP	65	31.40
Persistent glaucoma	84	40.58
Total	207	100

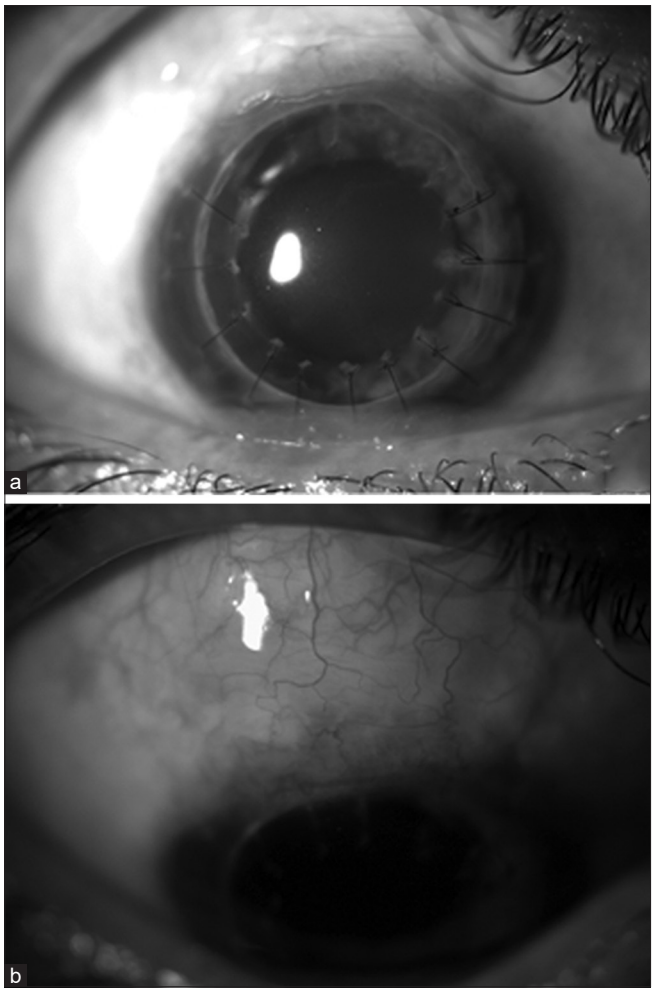

Figure 1: (a) Postoperative image of eye treated with optical penetrating keratoplasty for corneal scar. (b) Three months later, the patient underwent successful trabeculectomy surgery for post-PK glaucoma showing well-formed bleb. PK = penetrating keratoplasty

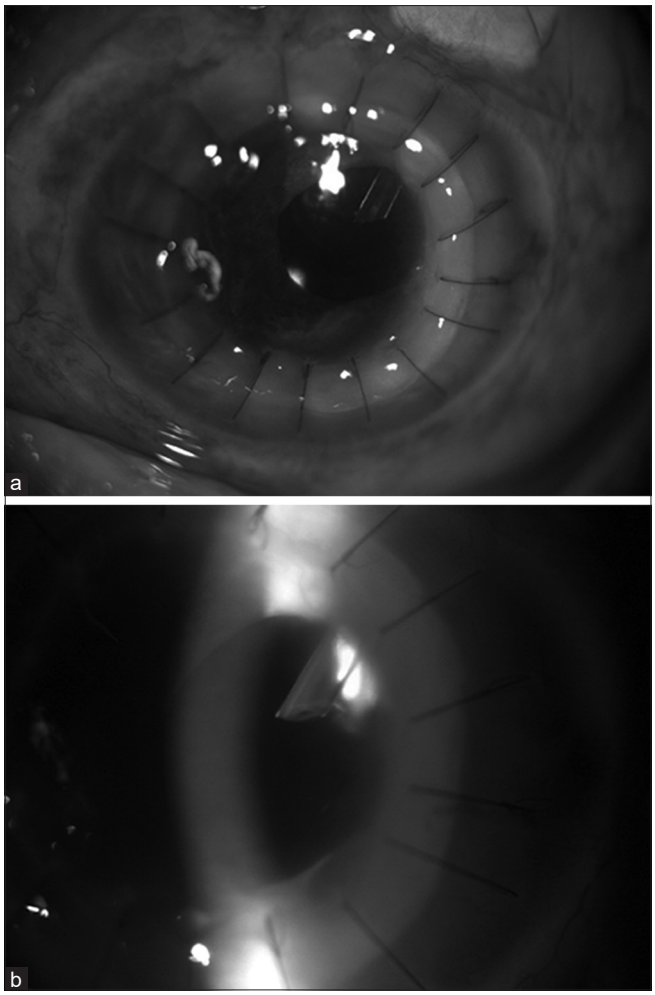

Figure 2: (a) Ahmed glaucoma valve (AGV) implantation in a patient with post-PK glaucoma. (b) AGV tube well placed in sulcus

transient rise in IOP. In univariate analysis odds of post-PK, persistent glaucoma was six times higher if the fellow eye had glaucoma (odds ratio: 6.03, 95% CI: 1.51–34.5). In regression analysis, it was found to be a significant factor (*P*=0.04). The graft–host disparity of more than 0.5 was found to be protective for developing post-PK glaucoma (*P*-value 0.04) in univariate analysis. Aphakia was present in 44 eyes, of which 30 eyes were aphakic preoperatively and postoperative aphakia was present in 14 (%) eyes. Out of 44 aphakic patients, persistent glaucoma developed in 20 (46%) eyes and it was a significant risk factor in monovariate analysis (*P*-value 0.03). Out of 92 pseudophakic patients, persistent glaucoma developed in 45 (48.9%). When this was compared with persistent glaucoma in phakic patients, the difference was found to be statistically significant (*P*-value 0.06). But there was no significant difference between aphakia and pseudophakia (*P*-value 0.7). Greater number of sutures (>16) was found to be a significant risk factor for developing post-PK glaucoma (*P*-value 0.049) in univariate analysis, but not in regression analysis. Among postoperative risk factors, presence of PAS was a significant factor in both univariate ($P < 0.001$) and multivariate (*P*-value 0.001) analyses.

The chances of developing persistent glaucoma increased with the number of risk factors present (preoperative, intraoperative, and postoperative combined). The odds of developing post-PK persistent glaucoma increased 3.24-fold, given that there was at least one risk factor present (compared to the base class of no risk factors present). The odds ratio increased to 3.81 with at least two risk factors, 4.17 with at least three risk factors, 4.60 with at least four risk factors, and 5.63 when there are at least five risk factors present. Logistic regression showed that the number of risk factors significantly affected the odds of developing post-PK persistent glaucoma and the odds ratio increased 1.20 times (*P*-value 0.011) for every additional risk factor. In 65 (89%) patients, IOP was controlled medically on topical glaucoma medical therapy alone. The mean number of AGM used was 2.5. IOP was not controlled in 19 patients despite maximum medical therapy. In these patients, surgical intervention was performed. Two patients underwent trabeculectomy, four patients had Ahmed glaucoma valve implantation [Fig. 2a and b], and two patients were treated with transscleral cyclophotocoagulation (TSCPC). In one patient, glaucoma was not controlled even after Ahmed glaucoma valve (AVG) implant and repeat glaucoma surgery was planned. The remaining 11 uncontrolled glaucoma

patients were planned for subsequent surgical intervention, which took place beyond the study period.

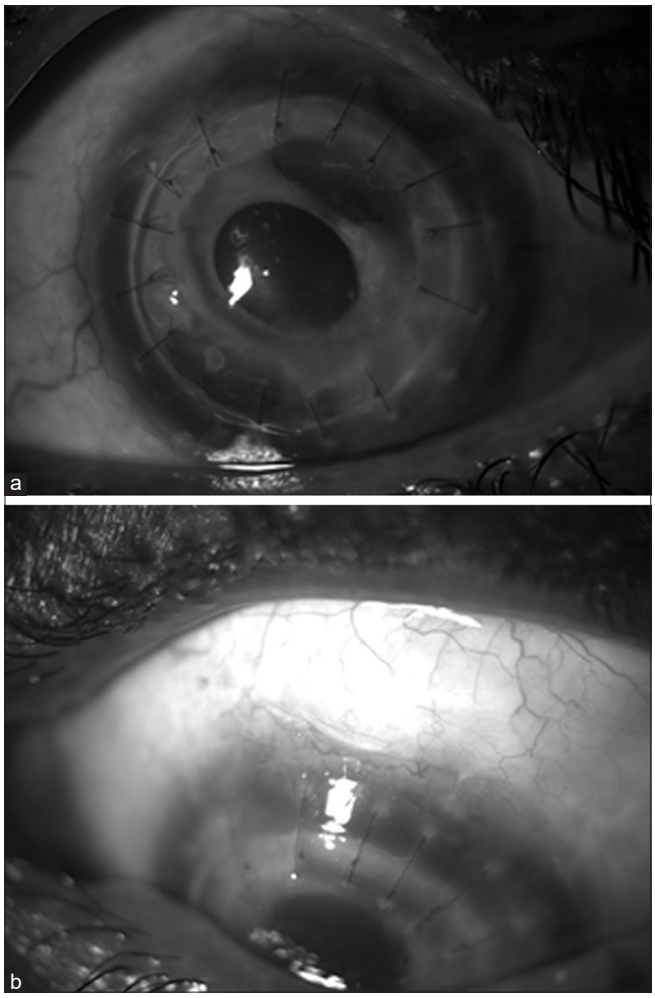

Figure 3: (a) Post-PK patient with 360° PAS developed secondary angle closure post-PK glaucoma. (b) Treated with trabeculectomy with Mitomycin-C (MMC), well-formed bleb can be seen

Discussion

Secondary ocular hypertension after PK is not uncommon. Intraocular pressure elevation can cause serious consequences after keratoplasty, in that it may lead to endothelial cell loss, early graft rejection, graft failure, and, over time, optic nerve damage. Evidence shows that corneal endothelium from grafted tissues is more susceptible to damage than healthy, nongrafted corneal endothelium. Hence, early detection of IOP elevation and its control are important to promote graft survival.[14]

In this study, IOP was measured by GAT wherever possible. DT was checked by experts and was taken as inferred IOP when GAT was not possible. Although newer methods of tonometry are available to measure IOP in these situations, these were not available with us in the period of study duration. Rubinfeld *et al.* suggest that for some patients, and for some surgeons, the finger tension or digital method of IOP estimation remains useful for detecting elevated IOP early after corneal transplantation.[15] In eyes with corneal pathology and after PK, an accurate and reliable estimation of IOP is often difficult due to corneal surface irregularities, corneal scars, high or irregular astigmatism, and corneal edema. Chen *et al.*[16] compared the utility of iCare, Tono-Pen, and noncontact airpuff tonometer with Goldmann applanation tonometer for measuring IOP in patients with corneal edema after PK. Poor agreement was noted between the Non contact tonometer (NCT) and GAT, as well as between the Tono-Pen and GAT, but the iCare showed clinically acceptable agreement with GAT. Rosentreter *et al.* found that in pathologic corneas, IOP was difficult to obtain with GAT and Dynamic contour tonometer (DCT), whereas Rebound tonometer (RT) was able to determine IOP in all pathologic corneas.[17]

The most common indication of PK in this study was corneal ulcer including perforated corneal ulcer (38.65%) [Table 5]. In a large-scale study, Dandona *et al.* analyzed 1,964 PKs at a tertiary eye care institute in India.[18] The most common indication for PK in their study was corneal scarring in 551 (28.1%) eyes, adherent leukoma in 147 (7.5%) eyes, regrafts in 336 (17.1%) eyes, and active infectious keratitis in 239 (12.2%) eyes.

Table 2: Indications of PK and development of persistent glaucoma

Indications	Number of cases found	Persistent post-PK glaucoma (number)	Persistent post-PK glaucoma (%)	95% CI
ABK	5	4	80	45%-100%
Failed graft	47	29	62	48%-76%
Corneal scar	37	16	43	27%-59%
Perforated corneal ulcer	42	14	33	19%-48%
Nonperforated corneal ulcer	38	11	29	15%-43%
Adherent leukoma	22	6	27	9%-46%
PBK	7	1	14	0%-40%
Congenital glaucoma	1	1	100	
Band-shaped keratopathy	1	1	100	
Corneal graft infection	1	1	100	
Corneal dystrophy	4	0	0	
Anterior staphyloma	1	0	0	
Peters' anomaly	1	0	0	

PK=penetrating keratoplasty

Table 3: Distribution of patients according to the presence of risk factors

Risk factors	Number of cases	Percentage of cases
Pre-op		
Shallow AC pre-op	92	44.44
Pre-op glaucoma	53	25.60
Repeat PK	47	22.71
Perforated corneal ulcer	42	20.29
Nonperforated corneal ulcer	38	18.36
Fellow eye glaucoma	14	6.76
Trauma	2	0.97
Intra-op		
Large graft	63	30.43
Combined surgery	57	27.54
Number of sutures	55	26.57
Vitrectomy	46	22.22
Same host recipient size	21	10.14
Bleeding interoperative	4	1.93
Post-op		
PAS	100	48.31
Increased inflammation in the early postoperative period	8	3.86
Hyphema	7	3.38
Aphakia	49	23.67
Endophthalmitis	6	2.90
Graft rejection	5	2.42
Total number of surgeries	207	

PK=Penetrating keratoplasty

The incidence of post-PK glaucoma varies from 5.3% to 60% across various studies. In our study, the incidence of persistent glaucoma was 40.5% (95% CI: 33.89–47.27). This is higher than the pooled estimate for the overall incidence of post PK glaucoma (PPKG), which was 21.5% (95% CI: 17.8–25.7) in a recent meta-analysis by Wu *et al*.[1] Ours is a tertiary eye care center; thus, large number of patients (198/207) belong to the high-risk category for the development of PPKG, such as those with perforated corneal ulcer, infective keratitis, repeat graft, vascularized scar, and aphakia. In a study by Sihota *et al*., the incidence of post-PK glaucoma was found to be 10.6% and preoperative corneal diagnosis of adherent leukomas was significantly associated with the development of postoperative glaucoma.[19] Huber *et al*. suggested that presence of preoperative glaucoma is an important risk factor for the development of post-PK glaucoma.[20]

Kirkness *et al*.[8] have postulated that repeat PK done for graft failure increases the risk of PPKG. Repeat PK increases the risk of PAS formation, and hence angle closure leading to post-PK glaucoma. Dada *et al*. highlighted that the main cause of late post-PK glaucoma is synechial angle closure, with the degree of synechial closure strongly correlating with the need for surgery. They suggested that a floppy, atrophic iris can lead to higher incidence of PAS formation, which can be prevented by iridoplasty.[21] In our study, the odds ratio for developing persistent glaucoma in the presence of repeat PK for failed graft was 3.08 (*P*-value 0.0008) in univariate analysis and the adjusted odds ratio for developing persistent glaucoma was 4.32 (*P*-value 0.007) in regression analysis.

Studies have reported that aphakic eyes are at a much higher risk of developing glaucoma.[5,21] The mechanism of glaucoma in aphakic cases that was proposed by Goldberg *et al*.,[2] Karesh *et al*.,[3] and Zimmerman *et al*.[22] was excessive intraocular manipulation leading to more inflammation. Angle distortion as a mechanism was described by Olson and Kaufman.[22,23] Mechanical collapse of the trabecular meshwork was proposed by Zimmerman *et al*.[2,3,22,24,25] Our study could not find aphakia as a significant risk factor in multivariate analysis. Kirkness *et al*. calculated the relative risk for the development of PPKG in the presence of PAS, which leads to secondary angle closure glaucoma. The extent of PAS by quadrants was evaluated and it was observed that the relative risk of development of glaucoma in association with PAS was 4 (95% CI: 2.7–5.2).[26] Similar to other studies, our study found PAS as a significant risk factor in both univariate and multivariate analyses [Fig. 3a and b].

Goldberg's study found that 71% of patients with preexisting glaucoma developed increased pressure in the early postoperative course.[2] Preoperative diagnosis of glaucoma in eyes with a corneal scar, corneal ulcer, or any media haze is difficult. In such a situation, we found that meticulous examination of the fellow eye and the presence of glaucomatous disk help in the suspicion of glaucoma in the eyes undergoing corneal transplant. An unadjusted odds ratio of 6.03 shows that the chances of developing glaucoma in PK eyes are high if the fellow eye glaucoma has been diagnosed preoperatively. The presence of fellow eye glaucoma resulting in a higher incidence of glaucoma in the eye being operated is a new factor to the best of our knowledge. This factor has not been studied yet, and further study with a larger sample size to support this finding can be considered. The terminology "fellow eye glaucoma" refers to glaucoma in the other eye, which was not diagnosed previously. The term preoperative glaucoma has been used for glaucoma in the same eye or in the fellow eye which was diagnosed earlier or if the patient is already on treatment for glaucoma.

Zimmerman *et al*. have shown that an oversized donor button (0.5 mm larger than the host bed) in aphakic eyes reduces the chance of post-PK glaucoma. The effect was more obvious when an 8-mm donor button was used in a 7.5-mm host bed.[22] However, Perl *et al*.[27] suggested that no additional benefit was seen on using oversized graft for post-PK glaucoma in any group (aphakia, pseudophakia, phakia). We found that the risk of developing persistent glaucoma is lesser when graft–host size disparity is >0.5 (*P*- 0.04). Many studies have found traumatized eyes and older patients to be at increased risk for developing glaucoma after PK.[2–5,7,28,29] In the present study, we found that patients above 50 years of age were more prone to develop post-PK glaucoma when compared with those <50 years of age and this difference was found to be significant in both univariate and multivariate analyses. Distribution of patients according to the presence of risk factors has been enumerated in Table 3. Other factors such as intraoperative bleeding and hyphema postoperatively and increased inflammation in the early postoperative period were responsible for causing a transient rise in IOP.

Table 4: Age, sex, and multivariate regression analysis for post-PK glaucoma

Risk factors	Number of incidence	Development of glaucoma					Unadjusted odds ratio		Multivariate adjusted odds ratio for developing glaucoma		Age, sex, and multivariate adjusted odds ratio for developing glaucoma		Multivariate adjusted odds ratio for developing persistent glaucoma		Age, sex, and multivariate adjusted odds ratio for developing persistent glaucoma		95% CI LL	95% CI UL
		No glaucoma	Early onset	Late onset	Transient	Persistent	Developing glaucoma	Developing persistent glaucoma	OR	*P*	OR	*P*	OR	*P*	OR	*P*		
Age											1.01	0.115			1.03	0.006	1.007	1.045
Sex											0.70	0.378			1.42	0.362	0.666	3.048
Aphakia	49	12	35	2	12	25	1.27	1.75	0.971	0.949	1.06	0.898	1.95	0.113	2.09	0.089	0.893	4.879
Bleeding intra-op	4	0	4	0	4	0	*	0.00	*	0.999	*	0.999	0.00	0.999	0.00	0.999	0.000	
Combined surgery	57	17	40	0	19	21	0.89	0.81	1.002	0.996	0.95	0.909	0.88	0.738	0.85	0.671	0.390	1.834
Endophthalmitis	6	0	6	0	3	3	*	1.48	*	0.999	*	0.999	0.71	0.738	0.69	0.707	0.100	4.771
Graft rejection	5	2	3	0	2	1	0.58	0.36	1.095	0.929	0.98	0.988	0.60	0.661	0.43	0.488	0.040	4.643
Hyphema	7	0	7	0	3	4	*	2.00	*	0.999	*	0.999	3.43	0.209	2.54	0.346	0.366	17.631
Increased inflammation in early post-op	8	0	8	0	5	3	*	0.87	*	0.999	*	0.999	0.47	0.384	0.58	0.542	0.104	3.287
Large graft	63	17	46	0	23	23	1.08	0.78	0.548	0.208	0.51	0.156	0.92	0.851	0.88	0.792	0.352	2.220
More number of sutures	55	7	48	0	20	28	3.46	1.78	2.854	0.029	2.77	0.036	1.75	0.152	1.63	0.225	0.740	3.605
Normal corneal ulcer	38	10	27	1	17	11	1.11	0.54	1.318	0.644	1.32	0.646	0.56	0.318	0.50	0.236	0.155	1.583
Fellow eye glaucoma	14	0	13	1	3	11	*	6.03	*	0.998	*	0.998	5.98	0.033	5.64	0.045	1.041	30.602
PAS	100	18	82	0	27	55	2.72	3.29	1.615	0.192	1.68	0.162	2.87	0.002	3.21	0.001	1.575	6.522
Perforated corneal ulcer	42	10	32	0	18	14	1.31	0.68	1.368	0.574	1.38	0.570	0.71	0.521	0.59	0.348	0.200	1.763
Pre-op glaucoma	53	7	45	1	15	31	3.25	2.69	2.777	0.056	2.47	0.097	1.14	0.772	0.99	0.991	0.401	2.468
Repeat PK	47	12	35	0	6	29	1.18	3.08	2.139	0.204	2.15	0.209	4.32	0.007	4.37	0.008	1.467	13.009
Same host recipient size	21	9	12	0	3	9	0.48	1.11	0.190	0.019	0.21	0.030	0.29	0.064	0.28	0.073	0.072	1.123
Shallow AC pre-op	92	21	71	0	34	37	1.60	0.97	1.197	0.656	1.20	0.652	0.94	0.859	1.03	0.933	0.486	2.193
Trauma	2	0	2	0	0	2	*	*	*	0.999	*	0.999	*	0.999	*	0.999	0.000	
Vitrectomy	46	12	34	0	14	20	1.13	1.17	0.975	0.955	1.04	0.938	0.89	0.787	0.92	0.851	0.387	2.189

LL=lower limit, OR=odds ratio, PK=penetrating keratoplasty, UL=upper limit, , *odd ratio could not be estimated as frequency is zero in few cells

Table 5: Distribution of the patients according to the indications of PK

Indications	Occurrence	Percentage	95% CI
Failed graft	47	22.71	(17%-28%)
Perforated corneal ulcer	42	20.29	(15%-26%)
Nonperforated corneal ulcer	38	18.36	(13%-24%)
Corneal scar	37	17.87	(13%-23%)
Adherent leukoma	22	10.63	(6%-15%)
PBK	7	3.38	(1%-6%)
ABK	5	2.42	(0%-5%)
Corneal dystrophy	4	1.93	(0%-4%)
Anterior staphyloma	1	0.48	(0%-1%)
Congenital glaucoma	1	0.48	(0%-1%)
Band-shaped keratopathy	1	0.48	(0%-1%)
Peters' anomaly	1	0.48	(0%-1%)
Corneal graft infection	1	0.48	(0%-1%)
Total	207	100.00	

PK=penetrating keratoplasty, ABK=Aphakic bullous keratopathy

Conclusion

Our study strongly recommends a meticulous examination of the fellow eye to assess the presence of glaucoma as it can help increase the suspicion of glaucoma in the eye to be operated. This can lead to developing an appropriate glaucoma management protocol for the eyes undergoing corneal transplant. Use of oversized graft (0.5 mm larger than the host bed) is recommended as it has been found to be protective for post-PK glaucoma.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Wu S, Xu J. Incidence and risk factors for post-penetrating keratoplasty glaucoma: A systematic review and meta-analysis. PloS One 2017;12:e0176261.
2. Goldberg DB, Schanzlin DJ, Brown SI. Incidence of increased intraocular pressure after keratoplasty. Am J Ophthalmol 1981;92:372–7.
3. Karesh JW, Nirankari VS. Factors associated with glaucoma after penetrating keratoplasty. Am J Ophthalmol 1983;96:160–4.
4. Chien AM, Schmidt CM, Cohen EJ, Rajpal RK, Sperber LT, Rapuano CJ, *et al*. Glaucoma in the immediate postoperative period after penetrating keratoplasty. Am J Ophthalmol 1993;115:711–4.
5. Wilson SE, Kaufman HE. Graft failure after penetrating keratoplasty. Surv Ophthalmol 1990;34:325–56.
6. Kirkness CM, Ficker LA. Risk factors for the development of postkeratoplasty glaucoma. Cornea 1992;11:427–32.
7. Foulks GN. Glaucoma associated with penetrating keratoplasty. Ophthalmology 1987;94:871–4.
8. Kirkness CM, Moshegov C. Post-keratoplasty glaucoma. Eye Lond Engl 1988;2(Suppl):S19-26.
9. Chaurasia S, Garg R, Beri S, Pakhare A. Sonographic assessment of optic disc cupping and its diagnostic performance in glaucoma. J Glaucoma 2019;28:131–8.
10. Ali NAM, Subrayan V, Reddy SC, Othman F. Comparison of B-scan sonographic measurements of optic cup with fundus photographic measurements. J Clin Ultrasound 2009;37:285–9.
11. Pujari A, Swamy DR, Selvan H, Agarwal D, Sihota R, Gupta S, *et al*. Clinical, ultrasonographic and optical coherence tomography correlation of optic nerve head cupping in glaucoma patients. Indian J Ophthalmol 2019;67:1663–6.
12. Sharma N, Sachdev R, Jhanji V, Titiyal JS, Vajpayee RB. Therapeutic keratoplasty for microbial keratitis. Curr Opin Ophthalmol 2010;21:293–300.
13. Sugar A, Sugar J. Techniques in penetrating keratoplasty: A quarter century of development. Cornea 2000;19:603–10.
14. Nguyen NX, Langenbucher A, Seitz B, Küchle M, Naumann GOH. Impact of increased intraocular pressure on long-term corneal endothelial cell density after penetrating keratoplasty. Ophthalmologica 2002;216:40–4.
15. Rubinfeld RS, Cohen EJ, Laibson PR, Arentsen JJ, Lugo M, Genvert GI. The accuracy of finger tension for estimating intraocular pressure after penetrating keratoplasty. Ophthalmic Surg Lasers 1998;29:213–5.
16. Yeh S-J, Chen K-H, Kuang T-M, Liu CJ-L, Chen M-J. Comparison of the iCare, Tono-Pen, non-contact airpuff, and Goldmann applanation tonometers in eyes with corneal edema after penetrating keratoplasty. J Chin Med Assoc 2021;84:320–5.
17. Rosentreter A, Athanasopoulos A, Schild AM, Lappas A, Cursiefen C, Dietlein TS. Rebound, applanation, and dynamic contour tonometry in pathologic corneas. Cornea 2013;32:313–8.
18. Dandona L, Ragu K, Janarthanan M, Naduvilath TJ, Shenoy R, Rao GN. Indications for penetrating keratoplasty in India. Indian J Ophthalmol 1997;45:163.
19. Sihota R, Sharma N, Panda A, Aggarwal HC, Singh R. Post-penetrating keratoplasty glaucoma: risk factors, management and visual outcome. Aust N Z J Ophthalmol 1998;26:305–9.
20. Huber KK, Maier A-KB, Klamann MKJ, Rottler J, Özlügedik S, Rosenbaum K, *et al*. Glaucoma in penetrating keratoplasty: Risk factors, management and outcome. Graefes Arch Clin Exp Ophthalmol 2013;251:105–16.
21. Dada T, Aggarwal A, Minudath KB, Vanathi M, Choudhary S, Gupta V, *et al*. Post-penetrating keratoplasty glaucoma. Indian J Ophthalmol 2008;56:269–77.
22. Zimmerman T, Olson R, Waltman S, Kaufman H. Transplant size and elevated intraocular pressure. Postkeratoplasty. Arch Ophthalmol 1978;96:2231–3.
23. Olson RJ. Aphakic keratoplasty. Determining donor tissue size to avoid elevated intraocular pressure. Arch Ophthalmol 1978;96:2274–6.
24. Olson RJ, Kaufman HE. A mathematical description of causative factors and prevention of elevated intraocular pressure after keratoplasty. Invest Ophthalmol Vis Sci 1977;16(12):1085–92.
25. Zimmerman TJ, Krupin T, Grodzki W, Waltman SR. The effect of suture depth on outflow facility in penetrating keratoplasty. Arch Ophthalmol 1978;96:505–6.
26. Kirkness CM, Ficker LA, Steele ADM, Rice NSC. The role of penetrating keratoplasty in the management of microbial keratitis. Eye 1991;5:425–31.
27. Perl T, Charlton KH, Binder PS. Disparate diameter grafting. Astigmatism, intraocular pressure, and visual acuity. Ophthalmology 1981;88:774–81.
28. Insler MS, Cooper HD, Kastl PR, Caldwell DR. Penetrating keratoplasty with trabeculectomy. Am J Ophthalmol 1985;100:593–5.
29. Irvine AR, Kaufman HE. Intraolar pressure following penetrating keratoplasty. Am J Ophthalmol 1969;68:835–44.